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FIBROUS GLASS AEROSOLS:

A literature Review

by

Bruce R. Laverty

Released by:

C. A. HARVEY, CAPT, MC, USN
Commanding Officer
Naval Submarine Medical Research Laboratory

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C. A. Harvey

C. A. HARVEY, CAPT, MC, USN
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ABSTRACT

Given the use of and exposure to fibrous glass lagging aboard submarines, a review of current scientific articles on the potential health hazards of fibrous glass was conducted. The review includes studies using animal, in vitro, and human data. No data were available on fibrous glass aerosol measurements aboard submarines. The majority of the studies reviewed did not deem human exposure to fibrous glass as a significant health hazard.

INTRODUCTION

The submarine atmosphere is a topic of interest, considering that once submerged, the craft relies on its own electrostatic precipitators (ESP's), scrubbers, and filters to create, ideally, an environment with minimal aerosolized toxic materials and other by-products. Historically, atmosphere sampling aboard nuclear submarines has shown cigarette smoke, lubricating oils, and cooking oils and fats to be the major contaminants (47,69,78,79). Other contaminants include: ozone, (major source: by-product of the ESP's); Freon,(major source: ship's refrigeration system and air conditioning plants); hydrogen, (major source: ship's batteries); carbon dioxide, (major source: human respiration); and carbon monoxide, (major source: cigarette smoking) (47). Contaminants tested for but not found were elemental mercury, and asbestos (47).

Considering that asbestos is no longer recommended for use, secondary to its carcinogenic and co-carcinogenic qualities, fibrous glass has become a common substitute. One use of fibrous glass aboard the Ohio class submarine is acoustic and thermal insulation around perforated ducting, which runs through many exposed, high traffic spaces, i.e. crew's berthing spaces. Although the raw fibrous glass is protected from the environment it is possible, through natural wear and tear of the housing material, that at some time the insulating material may become exposed and mechanically aerosolized. Obvious questions then are: a) do submarine aerosols contain fibrous glass, and b) are there health hazards related to the inhalation of these fibers?

FIBROUS GLASS AEROSOLS ABOARD SUBMARINES

To date no studies have been done to identify and quantify fibrous glass aerosols aboard submarines either through General Dynamics, Electric Boat Division, Groton, Connecticut or the Naval Submarine Medical Research Laboratory, Groton, Connecticut. Relatively few studies have been generated on the topic of fibrous glass aerosols in the medical literature. The remainder of this paper reviews our current knowledge as to the health hazard of exposure.

AEROSOLS. An aerosol is simply a solid or liquid particle suspended in a gas. The term includes both the liquid or solid, and the gas (40). Aerosol concentrations are expressed as either; 1.) mass concentration, which is the mass of the particulate in a unit volume of the aerosol, i.e. g/m^3, mg/m^3, ug/m^3, (this is the most common method), or 2.) number concentration, which is the number of particles per unit volume of aerosol, i.e. # of fibers/cc, mppcf (million particles per cubic foot).

FIBROUS GLASS. To satisfy the definition of fibrous glass two criteria must be met; 1.) a length-to-diameter ratio that equals or exceeds 3:1 and 2.) the composite of elements and ions in the fiber do not have a well ordered, regular structure with respect to each other. The fibers are glassy in appearance, not crystalline, as in asbestiform minerals, (40,42).

Historically fibrous glass was called mineral wool, which was a generic term for either slag wool or rock wool. Slag wool was produced by smelting and fiberizing molten iron ore, and was probably commercially done around 1885, in Manchester, England. Rock wool was produced by smelting and fiberizing naturally- occurring rocks and was first produced commercially in this country around 1900 in Alexandria, Indiana, (61). Using fibrous glass as thermal insulation in a private home was first accomplished by Owens-Corning company in 1933 in Lima, Ohio, (71). Processing and procedural adjustments of the molten glass has made fibers thinner and more consistent in diameter, which make modern fibrous glass what it is today.

Fibrous glass is of two forms, continuous filament and glass wool. Continuous filament, is used in textiles and fabrics, reinforcements in plastics, rubber, and paper, and has mean diameters between 6-10um. The second type is glass wool, and its most common use is for thermal and acoustic insulation. Diameter is important, as its thermal insulation effectiveness varies inversely with its diameter. Size varies from submicron to 10um in diameter, (42).

HEALTH HAZARDS OF FIBROUS GLASS AEROSOLS

The National Institute for Occupational Safety and Health, (NIOSH), first became interested in setting criteria for fibrous glass in 1968, when it had to address a list of 400-500 potentially harmful substances, of which fibrous glass was no. 40. In 1974 NIOSH held a symposium to bring together leading researchers in the field, in order to document what was presently known, and to identify on-going, or future research on the health hazards of fibrous glass. Based on the data available in 1977, NIOSH listed fibrous glass aerosols as a nuisance dust, putting it in the same category with paper dust, cotton dust, etc. setting exposure limits of 10 mg/m^3, based on an 8 hr/day/40 week exposure.

The same topics that were of interest in 1974 continue to present, and the data base has expanded. The carcinogenicity of asbestiform minerals is of great concern, especially when considering the increased use of fibrous glass. The long-term effects have continued to be studied. Fibrous tissue formation and carcinogenicity due to fibrous glass has been demonstrated in animal models after intrapleural and intraperitoneal implantation of fibrous glass, (Stanton and Wrench 1972, Wagner et al 1973, and Stanton 1977). Fiber diameter and length seem to determine its pathogenicity, with long thin fibers being the worst offenders. This phenomenon has been well studied and is believed to be related to the carcinogenecity of asbestos (36, 61, 90). Glass fibers mechanically induce damage rather than chemically. With increased use of fiber glass, often replacing asbestos, more needs to be known about its health effects. First and foremost, in order for fibrous glass to become a problem, it must first be inhaled, and to be inhaled, it must first be aerodynamically suitable.

LUNG DEPOSITION. The aerodynamic properties of fibers are best expressed as the aerodynamic diameter (D_{ae}). The D_{ae} is defined as the diameter of a unit density (1 g/cc) sphere that has the same settling velocity as the particle being studied, regardless of shape. Suffice it to say, the

physics behind this seemingly simple definition are beyond the scope of this paper, however, let it be understood that the aerodynamic diameter standardizes not only for shape, but also for density. So, the aerodynamic properties of a glass fiber with an aerodynamic diameter of 1um, (actual diameter of about .35um) will be the same as the aerodynamic properties of a droplet of water (density 1 g/cc) with a diameter of 1um. Aerodynamic properties are important for inhalation, but once inhaled, mechanisms of deposition become equally as paramount.

There are five mechanisms of particle deposition, four of which are important here. First is sedimentation, and it is proportional to the settling velocity of the particle and time available for settling. Next is impaction, which is determined by the inertial forces of the particle when a change in direction of airflow occurs, and it is also proportional to the settling velocity. Thirdly is Brownian displacement which is significant only for small particles in small airways. Finally interception, which occurs when the effective radius of a particle is less than the distance by which the center of the particle is separated from the airway surface. Deposition is then a function of linear dimensions (80). Respiratory architecture has a major influence on the mechanism of deposition, and location of deposition.

The respiratory architecture is made up of three regions. The head region consisting of the nose, mouth, throat, and larynx. The tracheobronchial region, includes the trachea, main stem bronchi, conducting, and terminal bronchioles. The final region is the alveolar region, and encompasses the respiratory bronchioles, alveolar ducts, and alveoli. The first two regions are lined with mucous and ciliated epithelium which carry deposited particles to the oropharynx, where the material is either swallowed, or coughed up and spat out. The alveolar region is cleansed by the work of alveolar macrophages. They engulf foreign particles, then transport them to the mucociliary escalator, or traverse the alveolar epithelium and deposit themselves in the parabronchial lymph nodes.

Considering the above background, fibers with slow falling velocities, (those with small aerodynamic diameters), that are inhaled must be retained within the lung to cause damage, if damage is going to occur. This means the fiber must be small enough to make it past the nasal hair filter, without being deposited in the tracheobronchial tree by impaction or interception, which would lead to removal of the fiber via the mucociliary escalator. If it can reach the alveolar region, it then has the chance to be deposited by sedimentation or Brownian diffusion. Most studies agree that a reasonable upper limit of fiber diameter, for a fiber to reach the alveolar region, is around 3.5um (or about a 10um aerodynamic diameter) (80). The question now arises, once deposited in the alveolar region does damage occur?

ANIMAL DATA ON DEPOSITION

Griffis et al, (30), used beagle dogs to determine the amount of inhaled radioactive glass fibers (mean diameter .15um and mean length 5.4um) that are deposited, after inhalation, in the alveolar region of the lung. The animals were monitored for four days post-inhalation. The dogs were then sacrificed

and radioactivity was totaled from the excreta, gastrointestinal tract, head, trachea, and lungs. From this it was determined that 11±6% of the total mass inhaled was deposited in the deep lung. Griffis' method was in good agreement with other studies, (30).

The location of deposition is likely going to be associated with sites of initial damage caused by inhaled fibers. Until recently, location of fiber deposition within the alveolar region has been based on mathematical formulation, (50). Brody and Roe, (12), demonstrated, using rats and hamsters, that inhaled particles small enough to reach the alveolar zone are deposited primarily at the alveolar bifurcation. The biological significance of this being there exists evidence that the initial lesions of asbestosis are a result of cellular responses at alveolar duct bifurcations. Whether this holds true for fibrous glass has yet to be reported, (12).

To document cellular and tissue changes, several investigators have tried intratracheal, intrapleural, and intraperitoneal injection of varying amounts and diameters of glass fibers into animal models, with varying results. Stanton and Wrench, (1972), Pott et al, (63), Stanton, (1977), and Pott et al, (1983), have produced pleural and peritoneal mesotheliomas by direct injection of large doses, (up to 40mg), of glass fibers with mean diameters less than 1um. Others, (17,24), have tried to reproduce these findings but have not been successful. Failure has been conjectured as secondary to smaller doses and inadequate time interval for experimental runs.

Inhalation studies, which more closely approximate the human aquisition of fibrous glass, have for the most part failed to produce significant lung pathology. Botham, (11), Gross, (31), and Schepers, (71) all used glass fibers with mean diameters less than 1 um in inhalation studies with mice, rats, hamsters, and monkeys. Results suggested a marked cellular response, i.e. macrophages containing glass fibers, and formation of giant cells, but no parenchymal fibrosis or carcinomata.

Bernstein et al, (6) varied the length and dose of the fibers in an inhalation study using rats. Three study groups were created in this assay; group I rats were exposed to an inhalant containing glass fibers with average dimensions of 1.5 um x 60 um given in a dose of 20 mg, group II rats received fibers with the average dimensions of 1.5 um x 5um given in a 2 mg dose, group III rats received the same fibers as group II rats except they were given in a 20 mg dose. These results also suggested a cellular response. Within groups II and III aggregations of glass fiber laden macrophages in the alveoli and lymph nodes were noticed. Except for a few small granulomata in group III, groups II and III only differed in magnitude of the response. The majority of fiber burden in group I was found in relatively large foreign body granulomata. No long fibers were noted in lymph nodes, suggesting lack of translocation.

To date animal studies have been carried out by either: 1.) artificially introducing glass fibers, (intratracheal, intrapleural, or intraperitoneal instillation), or by 2.) inhalation of aerosolized fibers. The results, i.e. amount of tissue damage, between these methods are a polar phenomena. Explanations for this vary. In a study by Pickrell et al, (62) both intratracheal instillation and inhalation of glass fibers was carried out

resulting in the expected dichotomous data, i.e. intratracheal instillation producing non-malignant lung damage, while the inhalation studies indicated minimal change. The authors suggested that even though the animals were exposed to large amounts, (50,000 fibers/cc) of aerosolized fibrous glass, the net amount inhaled was only about 3-5% of the lowest concentration of intratracheal instilled fibrous glass. Thus the difference in results of this study and others, may be related to actual lung burdens. Griffis et al, (29), suggests this same dose-response relationship to explain the difference in noted results between the two methodologies.

IN VITRO DATA. In vitro studies, (1,14,36,74,90), focus mainly on cytotoxicity of fibers to differing cell lines or cultures. In a study by Brown et al, (14), they concluded that fibrous glass was cytotoxic to Chinese Hamster V79-4 cells and a human tumor line of type II alveolar cells A549. Results suggested a threshold dose effect. Fibers with diameters less than 1.6 um and lengths greater than 10 um were cytotoxic as measured by malignant or premalignant changes in cell lines. Shorter and fatter fibers seemed to be inactive, however, Aalto et al, (1), noted just the opposite effect from small and fat fibers. They suggested that these fibers were capable of inducing macrophage fibrogenic factor (MMF). MMF is a substance produced by macrophages to stimulate functional activity of fibroblast, thereby leading to fibrosis. Other inducers include crystalline silica and asbestos.

Human bronchial cell cultures were used to determine cytotoxicity of four different fibers, one being fibrous glass (36). Results showed that fibrous glass was only mildly cytotoxic. Fibroblast cells from the same human donor were even more resistant to fibrous glass than the bronchial cells, showing no significant toxic effects. Though the authors offered no biologic significance to these findings, one can draw from the conclusions made concerning asbestos in this same study. Asbestos was found to be highly cytotoxic to human bronchial cells, and capable of stimulating abnormal cell growth patterns commonly observed in the pre-malignant human bronchus. It was demonstrated by electron microscopy that the asbestos fibers were phagocytized by the bronchial cells. Those cells that were introduced to larger fibers manifested more of the pre-malignant changes. Two mechanisms of action behind these changes are suggested: 1) mechanical induction of plasma membrane defects or 2) interaction between fiber and plasma membrane may lend to increased permeability of carcinogens, which would then induce changes. Perhaps since fibrous glass fragments along the horizontal axis (as opposed to the longitudinal axis like asbestos) resulting in relatively shorter fibers, the plasma membrane-fiber interaction is decreased. This could explain the lack of cytotoxicity of fibrous glass in this study.

HUMAN DATA. Human data consists to a large degree, of epidemiological and mortality studies. For the most part human exposure and animal inhalation data are in good agreement. Early reports, (83,59,32) have included written, radiologic, pulmonary function, and histologic surveys suggesting that exposures to fibrous glass dusts are not associated with harmful health effects. Some of these studies have been criticized for not quantitating the respirable fiber concentration, or not allowing adequate time after exposure to pick up latent pathology. More recent studies have been better designed to consider these important aspects.

In a study by Esman et al, (20), the concentration of fibers exposed to employees of 16 fibrous glass or mineral wool plants was quantified. Exposure was expressed as mass concentration, (mg/m^3), and fiber concentrations, (fibers/cc). Fiber counting for fiber concentration used both the optical and electron microscope. Data were displayed separately for both. The mass concentrations varied from less than .1 mg/m^3 to over 8mg/m^3 for the 16 plants. The median value of average exposure concentration was about 1.0 mg/m^3. The fiber concentration for all fibers ranged from .003 to more than 6 fibers/cc. The median value of average exposure concentration under the optical microscope was .028 fibers/cc, and for the electron microscope, (corresponding to submicron fibers), about .013 fiber/cc. It was determined that the submicron diameter fiber concentration was about 25% of total fiber concentration.

Using this data, Esman displayed cumulative distributions of airborne fiber diameters and determined that as fiber diameter increased in size, the percentage of airborne fibers would decrease. Three um was assumed to be the diameter of a respirable fiber, that is, upon inhalation by man a fiber 3 um or less can penetrate beyond the ciliated epithelium. Given this assumption, it was shown that 100% of the fiber burden with a nominal diameter of .07 um or less were respirable. Likewise, fibers 6 um and 10 um in nominal diameter, approximately 65% and 50% of the fiber burden, respectively, will be of a respirable diameter.

Morgan and Kaplan, (57), conducted a retrospective study of 6,536 employees engaged in fibrous glass production for 10 or more years. Results from this study suggested no statistically significant increased mortality for any cause of death, in particular no excess of fatal malignant or non-malignant respiratory diseases. Of this group, 1,272 individuals were employed 20 or more years and had experienced 30 or more years of latency at the time of the study. Separate analysis on this subgroup indicated no increased risk for lung cancer or chronic respiratory disease related to increased years of employment or time in latency.

There have been a handful of studies, (68,70), that have reported a significant excess of lung cancer deaths among employees of fibrous glass plants. However, Weill, (88), Enterline, (19), and others, (49,57) suggested confounding elements such as smoking history and previous occupational exposures were not taken into account nor controlled for.

Weill et al, (88), controlled for smoking history and age in a study of 1,028 employees of 7 fibrous glass or mineral wool plants, (median length of employment = 18 years). Results indicated no substantial lung disease in this cohort. Worker exposure was determined, and the highest concentrations were in a plant, (plant 5), producing a majority of microfibers. For fibers in the 1-3 um diameter range the median concentration was .210 fibers/cc, (all other plants < .032 fibers/cc). For fibers less than 1 um in diameter, the median concentration for plant 5 was .928 fibers/cc, (all other plants < .040 fibers/cc, except for plant 3 which had a median of < .203 fibers/cc).

Enterline et al, (19), in their mortality study wanted to compare results with a study by Robinson et al, (68), which suggested an excess in respiratory malignancies among fibrous glass workers exposed to fibers less

than 3 um. Enterline used a cohort of 12,851 employees of 17 glass fiber or mineral wool plants, with at least 1 year's exposure to fibers less than 3 um in diameter, between the years 1940 and 1963. Mean exposure to fibers less than 3 um was .039 fibers/cc for a mean of 11.17 years for fibrous glass workers, and a mean exposure of .353 fibers/cc for a mean of 11.08 years for mineral wool workers. The Robinson et al group used data on 1,448 of these same employees, (plant 9), for their study. The data produced by Enterline et al could not support the Robinson findings. Enterline went on to report that respiratory malignancies were not significantly in excess among the cohort under study, but there was an excess of non-malignant respiratory disease deaths. The authors included a caveat to the last finding admitting not to have controlled for smoking or previous work exposures, which in their opinion weaken this finding significantly.

SUMMARY. The obvious obstacle when studying health effects of environmental agents, such as fibrous glass, is the lengthy period of latency. The data on asbestos has typified this. In view of the asbestos story, it seems the damage is caused via its physical properties rather than its chemical properties, long thin fibers being the most harmful. Unlike asbestos, fiberglass does not fragment longitudinally into thinner and thinner fibers, rather it fragments horizontally into shorter fibers. The shorter and thicker fibers probably relate to the lack of pathogenicity of fibrous glass.

To date animal inhalation and human studies are in agreement, with no apparent long term, untoward health effects from fibrous glass aerosols. The support for the potential of injury comes from animal studies using artificial methods of instillation (intratracheal, intrapleural, intraperitoneal) of large doses of glass fibers, producing pleural and peritoneal mesotheliomas. As Pickrell et al pointed out, animal inhalation studies using exposure concentrations of 50,000 fibers/cc are only 3-5% of the lowest intratracheal installation concentration. The human exposure concentrations collected from the data reviewed, showed exposures from .003 - 6 fibers/cc ! Realizing the orders of magnitude difference between animal exposures that produce cancers and actual human exposures make it difficult to translate from one model to the other.

The in vitro data to date adds little enlightenment either way given the difficulty of extrapolating data to the human condition. Its greatest asset will probably lie in its ability to demonstrate mechanisms of injury.

RECOMMENDATION

Published literature to date suggests that human exposure to fibrous glass aerosols is not a significant health hazard, especially regarding any increase in malignant and non-malignant respiratory disease. This appears even more evident after controlling for age and smoking history. It is recommended however, (46,49), that high standards for use be maintained, including minimal airborne exposure, respiratory protection as well as eye and skin protection, so that our current lack of scientific proof does not lead to a false sense of complacency, as we once were in regard to asbestos.

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Given the use of and exposure to fibrous glass lagging aboard submarines, a review of current scientific articles on the potential health hazards of fibrous glass was conducted. The review includes studies using animal, in vitro, and human data. No data were available on fibrous glass aerosol measurements aboard submarines. The majority of the studies reviewed did not deem human exposure to fibrous glass as a significant health hazard.

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